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Chronic Hypertension in Pregnancy and Racial–Ethnic Disparities in Complications

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OBJECTIVE: To evaluate whether there are individual- and population-level associations between chronic hypertension and pregnancy complications, and to assess differences across seven racial–ethnic groups.

METHODS: This population-based study used linked vital statistics and hospitalization discharge data from all live and stillbirths in California (2008–2018), Michigan (2008–2020), Oregon (2008–2020), Pennsylvania (2008–2014), and South Carolina (2008–2020). We used multivariable log-binomial regression models to estimate risk ratios (RRs) and population attributable risk (PAR) percentages with 95% CIs for associations between chronic hypertension and several obstetric and neonatal outcomes, selected based on prior evidence and pathologic pathways. We adjusted models for demographic factors (race and ethnicity, payment method, educational attainment), age, body mass index, obstetric history, delivery year, and state, and conducted analyses stratified across seven racial–ethnic groups.

RESULTS: The study included 7,955,713 pregnancies, of which 168,972 (2.1%) were complicated by chronic hypertension. Chronic hypertension was associated with several adverse obstetric and neonatal outcomes, with the largest adjusted PAR percentages observed for preeclampsia with severe features or eclampsia (22.4; 95% CI 22.2–22.6), acute renal failure (13.6; 95% CI 12.6–14.6), and pulmonary edema (10.7; 95% CI 8.9–12.6). Estimated RRs overall were similar across racial–ethnic groups, but PAR percentages varied. The adjusted PAR percentages (95% CI) for severe maternal morbidity—a widely used composite of acute severe events—for people who were American Indian or Alaska Native, Asian, Black, Latino, Native Hawaiian or Other Pacific Islander, White, and Multiracial or Other were 5.0 (1.1–8.8), 3.7 (3.0–4.3), 9.0 (8.2–9.8), 3.9 (3.6–4.3), 11.6 (6.4–16.5), 3.2 (2.9–3.5), and 5.5 (4.2–6.9), respectively.

CONCLUSION: Chronic hypertension accounts for a substantial fraction of obstetric and neonatal morbidity and contributes to higher complication rates, particularly for people who are Black or Native Hawaiian or Other Pacific Islander.

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Chronic hypertension affects approximately 85,000 births (2.3%) in the United States each year.[1] The prevalence of chronic hypertension among pregnant and reproductive-aged people has been steadily increasing over time, as has the mortality rate due to complications associated with chronic hypertension in pregnancy.[2–4] Obstetric complications can include

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superimposed preeclampsia, eclampsia, cerebrovascular accidents, placental abruption, acute renal failure, pulmonary edema, and postpartum hemorrhage.[4–8] Additionally, chronic hypertension has been associated with higher incidence of fetal and neonatal complications, including stillbirth, fetal growth restriction, congenital anomalies, preterm birth, and low birth weight.[4–9] However, these prior findings are derived from births in the 1990s or early 2000s or from small hospital cohorts, which may not reflect current patient populations or clinical practices. Further, the individual-level relative risks reported in these studies have limited utility in understanding the effect of chronic hypertension during pregnancy in the U.S. population and subpopulations.[4,6–9]

National surveillance data have shown that chronic hypertension affects a higher percentage of reproductive-age women who are Black (15–19%) than those who are White (6–10%) or Latino (3–10%).[3,10] A report in 2022 from the Centers of Disease Control and Prevention further found that cardiovascular conditions, hypertensive disorders of pregnancy, and cerebrovascular accidents were the underlying cause of death in a higher percentage of pregnancy-related deaths in Black people (66%) than in other racial–ethnic groups assessed (Asian, Latino, White).[11] However, the lack of race and ethnicity information in routinely collected administrative health data has been a barrier to measuring racial–ethnic disparities in chronic hypertension during pregnancy and the potential effect on disparities in obstetric and neonatal complications.[4,12]

Our objective for this study was to evaluate the hypotheses that chronic hypertension is associated with birth complication rates on both the individual and population levels, and that differences exist in these associations across seven racial–ethnic groupings in multi-state, linked vital statistics and hospitalization discharge data.

METHODS

We conducted a population-based study of live- and stillbirths in California (2008–2018), Michigan (2008–2020), Oregon (2008–2020), Pennsylvania (2008–2014), and South Carolina (2008–2020). Live birth and fetal death certificate data were previously linked to obstetric and neonatal discharge data from birth hospitalizations using probabilistic linkage techniques.[13] This study was part of a larger, ongoing project to build a repository of state-level data sets of linked vital statistics and hospitalization discharge data. At the time of analysis, data from five states (California, Michigan, Oregon, Pennsylvania, and South Carolina) were available. States differ in when they began linking data sets and in the most recent year of data linked. We included all available data beginning in 2008, which is the first year that all five states had implemented the U.S. Revised Certificates of Live Birth and Fetal Death. We included births with linked records and complete information on variables used in study analysis. Births with gestational ages less than 23 weeks or more than 42 weeks were excluded.[14] As shown in Figure 1, we used a full neonatal sample (including multifetal gestations) for analyses of neonatal outcomes. For analyses of obstetric outcomes, we excluded duplicate obstetric patient records in cases of multifetal gestation. That is, if an obstetric patient had a multifetal gestation and an obstetric complication, they only counted as one case of the obstetric complication. The Stanford University Research Compliance Office and the state institutional review boards provided ethics approval. We followed the RECORD (REporting of studies Conducted using Observational Routinely-collected Data) guidelines for the reporting of this observational cohort study using routinely collected health data.

Chronic hypertension was the exposure of interest in analyses. We used International Classification of Diseases, Ninth and Tenth Revisions, Clinical Modification (ICD-9-CM and ICD-10-CM) diagnosis codes from the birth hospitalization to identify people with chronic hypertension. Appendix 1, available online at http://links.lww.com/AOG/D364, provides the data source

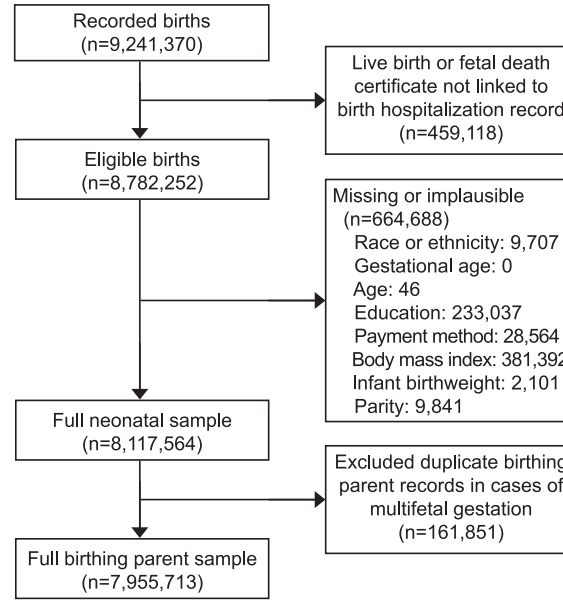

Fig. 1. Study sample selection.
Leonard. Chronic Hypertension in Pregnancy. Obstet Gynecol 2023.

and ICD-9-CM and ICD-10-CM codes for each variable used in this study. We selected outcomes for analysis based on prior evidence and physiologic plausibility.[3–9,15,16] The obstetric outcomes assessed included: preeclampsia with severe features or eclampsia, mode of birth, placental abruption, postpartum hemorrhage, cerebrovascular accident, pulmonary edema, acute renal failure, and severe obstetric morbidity. Preeclampsia superimposed on chronic hypertension is widely considered to have clinical severity akin to preeclampsia with severe features. Therefore, individuals with chronic hypertension were not at risk of gestational hypertension or preeclampsia without severe features, and we could not examine these outcomes. Further, ICD-CM diagnosis codes for preeclampsia superimposed on chronic hypertension do not specify severity. For mode of birth, vaginal births with vacuum or forceps assistance were categorized as operative vaginal births; other vaginal births were categorized as spontaneous vaginal births. Cesarean births after either induction of labor or a labor complication were categorized as unplanned cesarean birth; all other cesarean births were categorized as planned cesarean births, with recognition of labor-based categorization as an imperfect proxy for unplanned and planned cesarean birth. We separated cesarean birth because a prior study reported higher odds of planned, but not unplanned, cesarean birth among people with chronic hypertension.[8] Severe obstetric morbidity was a composite outcome of 20 indicators defined by the Centers for Disease Control and Prevention and the Maternal and Child Health Bureau.[17]

The neonatal outcomes that we assessed included stillbirth, small for gestational age, gestational age at birth, birth weight, and congenital anomalies. *Stillbirth* was identified using Certificates of Fetal Death and is defined as fetal death at or after 20 weeks of gestation in California, at or after 20 weeks of gestation or birth weight of at least 400 g in Michigan, at or after 16 weeks of gestation in Pennsylvania, and at or after 20 weeks of gestation or birth weight of at least 350 g in Oregon and South Carolina.[18] *Small for gestational age* was defined as birth weight less than the 10th percentile for gestational age and sex using U.S. reference charts.[19] Gestational age at birth was categorized as 23–27 weeks (extremely preterm), 28–31 weeks (very preterm), 32–36 weeks (late preterm), and 37–42 weeks (term).[20] Birth weight was categorized as less than 1,500 g (very low birth weight), 1,500–2,499 g (low birth weight), and 2,500 g or greater (not low birth weight).[21] Congenital anomalies were separated into cardiovascular anomalies and other anomalies because a prior study reported an association between chronic hypertension and cardiac, but not other, anomalies.[9] We included a broad set of congenital anomalies likely to affect neonatal health, as determined by the Joint Commission quality measure for newborn outcomes.[22]

Covariates used in analyses were selected a priori based on directed acyclic graphs, prior evidence, and available data.[2–9] These covariates included patient age, social demographic factors (educational attainment, payment method for birth hospitalization, race and ethnicity), obstetric history (nulliparous, multiparous without prior cesarean birth, or multiparous with prior cesarean birth), prepregnancy body mass index (BMI, calculated as weight in kilograms divided by height in meters squared), delivery year, and state (however, the South Carolina data did not specify whether BMI data in vital statistics were prepregnancy). Additionally, we descriptively assessed comorbidities related to chronic hypertension, including chronic renal disease, preexisting diabetes, preexisting cardiac disease, and thyroid disorder. We did not include these or other conditions that could be on the causal pathway between chronic hypertension and the outcomes of interest (eg, gestational diabetes mellitus). The Centers for Disease Control and Prevention instructs that race and ethnicity be self-reported on live birth and fetal death certificates. State health departments categorize responses to align with the U.S. Office of Management and Budget standards on race and ethnicity classifications.[23] These categories include Latino (Latino, Hispanic, or Spanish origin) and, for non-Latino respondents, American Indian or Alaska Native, Asian, Black (Black or African American), Native Hawaiian or Other Pacific Islander, White, and Multiracial or Other. State health departments previously categorized specified races not otherwise classified above as Other in the available data. People without race and ethnicity data were excluded from analyses.

For the statistical analysis, we first calculated the distribution of patient characteristics by chronic hypertension status and the prevalence of chronic hypertension by race and ethnicity. We then evaluated the associations between chronic hypertension and the outcomes using multivariable generalized linear models to estimate risk ratios (RRs) and population attributable risk (PAR) percentages with 95% CIs. We used a Poisson distribution, log link, and robust standard errors in the models, and adjusted for age, educational attainment, payment method for birth hospitalization, race and ethnicity, BMI, obstetric history, delivery year, and state. In the neonatal outcome models, robust standard errors accounted for correlation between neonates of multifetal gestations.[24] For outcomes with three or more categories,

we performed a series of models with the same reference outcome (eg, each delivery mode outcome was compared with spontaneous vaginal birth). We calculated PAR percentages as a way to estimate the population-level effect of chronic hypertension on the outcomes. The PAR percentage is defined as the percentage of all outcome events that is attributable to a given exposure and is equivalent to: $100 \times \left(\frac{\textit{Observed incidence} - \textit{Expected incidence if exposure eliminated}}{\textit{Observed incidence}}\right)$. Obstetric outcomes were analyzed among birthing parents (total N=7,955,713), and neonatal outcomes were analyzed among neonates (total N=8,117,564). All models were conducted in the full study population and stratified by racial–ethnic group. Statistical analyses were performed in Stata 17, and we calculated PAR percentages with CIs using the PUNAF postestimation command.[25]

RESULTS

The study included 7,955,713 people giving birth, of whom 168,972 (2.1%) had chronic hypertension (Fig. 1). By racial–ethnic group, the prevalence of chronic hypertension was 2.5% in American Indian or Alaska Native, 1.6% in Asian, 5.1% in Black, 1.5% in Latino, 2.9% in Native Hawaiian or Other Pacific Islander, 2.0% in White, and 2.3% in Multiracial or Other people. Compared with people without chronic hypertension, a higher proportion of people with chronic hypertension were 35 years or older, Black, and multiparous with prior cesarean birth (Table 1). The prevalence of obesity, chronic renal disease, preexisting diabetes, preexisting cardiac disease, and thyroid disorder were notably higher in people with chronic hypertension than in others.

Chronic hypertension was associated with increased risk of all outcomes assessed, except for operative vaginal birth (Table 2). The highest adjusted risk ratios (aRRs) and PAR percentages were observed for preeclampsia with severe features or eclampsia (aRR 10.4, 95% CI 10.3–10.5; adjusted PAR percentage 22.4, 95% CI 22.2–22.6), acute renal failure (aRR 5.85, 95% CI 5.39–6.36; adjusted PAR percentage 13.6, 95% CI 12.6–14.6), and pulmonary edema (aRR 4.86, 95% CI 4.08–5.80; adjusted PAR percentage 10.7, 95% CI 8.9–12.6). As an example, chronic hypertension was associated with 5.85 times the risk of acute renal failure (95% CI 5.39–6.36), compared with no chronic hypertension, and was estimated to contribute to 13.6% (95% CI 12.6–14.6) of acute renal failure cases in the study population, independent of measured confounders.

Adjusted risk ratios for obstetric and neonatal outcomes associated with chronic hypertension tended to be similar across racial–ethnic groups (Tables 3 and 4). However, some differences included the RR for preeclampsia with severe features or eclampsia being higher for Asian people (aRR 15.67, 95% CI 15.01–16.37), the RR for acute renal failure being higher for Latino people (aRR 9.82, 95% CI 8.44–11.43), and the RRs for acute renal failure (aRR 9.03, 95% CI 4.20–19.43), stillbirth (aRR 3.77, 95% CI 1.75–8.12), and extremely preterm birth (aRR 7.13, 95% CI 4.19–12.11) being higher for Native Hawaiian or Other Pacific Islander people.

Estimated PAR percentages across racial–ethnic groups represented the percentage of cases for each outcome that was attributable in part to chronic hypertension in the given racial–ethnic group (Tables 5 and 6). Among obstetric outcomes, PAR percentages for severe obstetric morbidity and acute renal failure were highest in Black and Native Hawaiian or Other Pacific Islander people and lowest in White people. The PAR percentage for cerebrovascular accident was also highest in Black people (and could not be estimated in Native Hawaiian or Other Pacific Islander people due to small sample size). Among neonatal outcomes, PAR percentages for stillbirth, extremely preterm birth, and very low birth weight were highest in Black and Native Hawaiian or Other Pacific Islander and lowest in White and Latino people. As an example interpretation, it was estimated that chronic hypertension contributed to 9.0% (95% CI 8.2–9.8) of severe obstetric morbidity cases in Black people, 11.6% (95% CI 6.4–16.5) in Native Hawaiian or Other Pacific Islander people, and 3.2% (95% CI 2.9–3.5) in White people, independent of measured confounders.

DISCUSSION

In this multi-state, population-based study, we found that chronic hypertension in pregnancy was associated with substantially higher risk of adverse obstetric and neonatal outcomes across seven racial–ethnic groups. Additionally, chronic hypertension was estimated to contribute to a considerable percentage (5–22%) of cases of preeclampsia with severe features or eclampsia, cerebrovascular accidents, acute renal failure, severe obstetric morbidity, very preterm birth (gestational age less than 32 weeks), and very low birth weight (less than 1,500 g). The relative risk of outcomes associated with chronic hypertension in pregnancy were generally similar in magnitude across racial–ethnic groups. However, PAR percentages indicated that chronic hypertension contributed to a

Table 1. Characteristics of People Giving Birth, by Chronic Hypertension Status

Characteristic	Chronic Hypertension	
	No (n=7,786,741)	Yes (n=168,972)
Age (y)	28±6	32±6
Category		
Younger than 25	2,185,622 (28.1)	20,745 (12.3)
25–34	4,273,529 (54.9)	90,584 (53.6)
35 or older	1,327,590 (17.1)	57,643 (34.1)
Race and ethnicity		
American Indian or Alaska Native	28,137 (0.4)	733 (0.4)
Asian	748,783 (9.6)	11,846 (7.0)
Black	751,814 (9.7)	40,544 (24.0)
Latino	2,640,098 (33.9)	41,016 (24.3)
Native Hawaiian or Other Pacific Islander	22,123 (0.3)	657 (0.4)
White	3,378,731 (43.4)	69,031 (40.9)
Multiracial or Other*	217,055 (2.8)	5,145 (3.0)
Educational attainment		
Less than high school degree	1,346,424 (17.3)	22,507 (13.3)
High school degree or equivalent	1,993,527 (25.6)	44,108 (26.1)
Some college	2,177,601 (28.0)	58,364 (34.5)
Undergraduate degree or higher	2,269,189 (29.1)	43,993 (26.0)
Method of payment for birth		
Commercial insurance	3,944,450 (50.7)	89,486 (53.0)
Government insurance	3,619,448 (46.5)	76,529 (45.3)
Self-pay or other	222,843 (2.9)	2,957 (1.8)
Obstetric history		
Nulliparous	3,099,876 (39.8)	57,117 (33.8)
Multiparous without prior cesarean	3,375,340 (43.4)	66,791 (39.5)
Multiparous with prior cesarean	1,311,525 (16.8)	45,064 (26.7)
Prepregnancy BMI (kg/m^2)†	26±6	34±9
Category		
Underweight (lower than 18.5)	299,429 (3.9)	1,199 (0.7)
Normal weight (18.5–24.9)	3,663,646 (47.1)	27,887 (16.5)
Overweight (25–29.9)	2,008,124 (25.8)	36,959 (21.9)
Obesity class 1 (30–34.9)	1,042,833 (13.4)	37,346 (22.1)
Obesity class 2 (35–39.9)	471,286 (6.1)	28,794 (17.0)
Obesity class 3 (40 or higher)	301,423 (3.9)	36,787 (21.8)
Chronic renal disease	17,836 (0.2)	3,442 (2.0)
Preexisting diabetes	72,095 (0.9)	15,224 (9.0)
Preexisting cardiac disease	58,034 (0.8)	6,349 (3.8)
Thyroid disorder	238,198 (3.1)	11,359 (6.7)

Data are mean±SD or n (%).
BMI, body mass index.
* Includes people who identified as multiple races or as a race not classified with the other racial groups, defined previously in the data sets by state health departments.
† Prepregnancy or at delivery not specified in South Carolina BMI variable data.

higher proportion of adverse outcomes among Black and Native Hawaiian or Other Pacific Islander people than among other people. These differences were driven in part by higher prevalence of chronic hypertension in Black and Native Hawaiian or Other Pacific Islander people (5.1% and 2.9%, respectively, vs 2.1% overall).

Our study showed how disparities in chronic hypertension across several racial–ethnic groups likely contribute to racial inequities in obstetric and neonatal outcomes. The lack of race and ethnicity information in some large hospitalization discharge data sets has precluded the ability to evaluate how chronic hypertension is associated with disparities in uncommon outcomes, such as severe obstetric morbidity.[4,12] Further, prior studies on racial–ethnic disparities in chronic hypertension rates have largely categorized race and ethnicity as Black, Latino, White, and Other, despite people categorized in the Other group representing a large, diverse, and growing proportion of the U.S. population.[3,10] A study of California births from 2011 to 2017 found the highest rates of severe obstetric morbidity to be 2.5% in

Table 2. Associations Between Chronic Hypertension and Outcomes in the Full Study Population

Outcome	Crude RR (95% CI)	Adjusted RR (95% CI)*	Adjusted PAR (%) (95% CI)*
Obstetric			
Preeclampsia with severe features or eclampsia	15.2 (15.1–15.4)	10.4 (10.3–10.5)	22.4 (22.2–22.6)
Mode of birth†			
Operative vaginal	0.88 (0.85–0.91)	0.98 (0.95–1.01)	−0.03 (−0.07 to 0.01)
Unplanned cesarean	1.89 (1.88–1.91)	1.27 (1.26–1.28)	0.7 (0.7–0.8)
Planned cesarean	1.91 (1.89–1.92)	1.19 (1.18–1.19)	0.6 (0.5–0.6)
Placental abruption	1.83 (1.76–1.90)	1.78 (1.71–1.84)	1.7 (1.5–1.8)
Postpartum hemorrhage	1.54 (1.50–1.57)	1.45 (1.42–1.48)	1.0 (0.9–1.1)
Cerebrovascular accident	6.24 (5.71–6.81)	3.34 (3.02–3.69)	8.4 (7.4–9.3)
Pulmonary edema	7.21 (6.21–8.37)	4.86 (4.08–5.80)	10.7 (8.9–12.6)
Acute renal failure	9.08 (8.48–9.72)	5.85 (5.39–6.36)	13.6 (12.6–14.6)
Severe obstetric morbidity‡	3.62 (3.51–3.74)	2.66 (2.57–2.75)	4.5 (4.3–4.8)
Neonatal			
Stillbirth	2.66 (2.45–2.90)	1.98 (1.81–2.17)	2.7 (2.3–3.2)
SGA (birth weight less than the 10th percentile)	1.44 (1.43–1.46)	1.56 (1.54–1.58)	1.1 (1.1–1.1)
Gestational age at birth (wk)†			
23–27	4.80 (4.63–4.98)	3.41 (3.27–3.55)	5.8 (5.5–6.0)
28–31	5.22 (5.09–5.36)	4.29 (4.17–4.41)	6.9 (6.6–7.1)
32–36	2.73 (2.71–2.76)	2.38 (2.35–2.40)	3.2 (3.1–3.2)
Birth weight (g)†			
Less than 1,500	5.43 (5.32–5.55)	4.21 (4.11–4.31)	7.4 (7.2–7.6)
1,500–2,499	2.66 (2.63–2.69)	2.50 (2.47–2.53)	3.2 (3.1–3.2)
Congenital anomaly†			
Cardiovascular	2.53 (2.47–2.59)	1.99 (1.95–2.04)	2.6 (2.5–2.7)
Other anomaly	1.55 (1.51–1.58)	1.37 (1.34–1.40)	0.9 (0.8–0.9)

RR, risk ratio; PAR, population attributable risk; SGA, small for gestational age.

* Multivariable models include age, educational attainment, payment method for birth hospitalization, race and ethnicity, body mass index, obstetric history, delivery year, and state.

† For multinomial outcomes, each outcome was compared with the most common outcome (spontaneous vaginal birth, gestational age 37–42 weeks, birth weight 2,500 g or greater, and no congenital anomaly).

‡ Composite outcome of 20 indicators defined by the Centers for Disease Control and Prevention and the Maternal and Child Health Bureau.[17]

Black people, 2.2% in Native Hawaiian or Other Pacific Islander people, and 2.1% in American Indian or Alaska Native people.[26] In our study, we found the highest rates of chronic hypertension to be 5.1% in Black people, 2.9% in Native Hawaiian or Other Pacific Islander people, and 2.5% in American Indian or Alaska Native people. Relatedly, we found evidence that chronic hypertension contributes to a higher proportion of severe obstetric morbidity and acute renal failure among Black and Native Hawaiian or Other Pacific Islander people than among other racial–ethnic groups. A higher proportion of other severe obstetric complications–cerebrovascular accidents and pulmonary edema–was also attributable to chronic hypertension among Black people, and these complications were too rare to assess among American Indian or Alaska Native and Native Hawaiian or Other Pacific Islander people. The results further showed chronic hypertension to be a particularly strong contributor to severe fetal and neonatal complications among Black and Native Hawaiian or Other Pacific Islander people, although CIs tended to be large for Native Hawaiian or Other Pacific Islander and American Indian or Alaska Native people due to sample size limitations. These outcomes included stillbirth, very preterm birth (less than 32 weeks of gestation), and very low birth weight (less than 1,500 g). Our findings support the disaggregation of race and ethnicity data in the United States, particularly for Indigenous and Pacific Islander people,[27] to identify and prevent disparities in obstetric and neonatal health.

The study findings also contribute updated evidence on the effects of chronic hypertension on birth outcomes among approximately 8 million births from five demographically and geographically diverse states in recent years. The relative risks of outcomes associated with chronic hypertension in pregnancy largely corroborate those from older cohort studies.[4,6,9] The large sample size and linkage of vital statistics and hospitalization discharge data in this study allowed granular examination of several outcomes for both the birthing parent and for the neonate, such as mode of birth, gestational age, and congenital anomalies, and statistical adjustment for demographic and clinical factors, including BMI. We found chronic hypertension to be similarly associated

Table 3. Adjusted Risk Ratios* and 95% CIs for Associations Between Chronic Hypertension and Obstetric Outcomes, Stratified by Racial–Ethnic Group

Outcome	American Indian or Alaska Native	Asian	Black
Preeclampsia with severe features or eclampsia	10.14 (8.57–12.00)	15.67 (15.01–16.37)	7.73 (7.55–7.92)
Mode of birth‡			
Operative vaginal	0.53 (0.24–1.20)	1.04 (0.96–1.14)	0.89 (0.82–0.96)
Unplanned cesarean	1.22 (1.09–1.38)	1.31 (1.28–1.35)	1.26 (1.24–1.28)
Planned cesarean	1.24 (1.14–1.34)	1.26 (1.23–1.29)	1.17 (1.15–1.18)
Placental abruption	1.30 (0.68–2.48)	1.80 (1.58–2.06)	1.71 (1.58–1.84)
Postpartum hemorrhage	1.07 (0.77–1.48)	1.50 (1.41–1.60)	1.48 (1.42–1.55)
Cerebrovascular accident	—§	6.04 (3.59–10.16)	3.19 (2.71–3.74)
Pulmonary edema	—§	4.09 (2.41–6.92)	3.44 (2.38–4.99)
Acute renal failure	—§	6.36 (4.85–8.34)	4.72 (4.07–5.47)
Severe obstetric morbidity	2.53 (1.52–4.22)	2.86 (2.53–3.23)	2.56 (2.40–2.73)

Outcome	Latino	Native Hawaiian or Other Pacific Islander	White	Multiracial or Other†
Preeclampsia with severe features or eclampsia	12.74 (12.47–13.01)	9.65 (8.19–11.36)	9.78 (9.59–9.97)	10.98 (10.29–11.71)
Mode of birth‡				
Operative vaginal	0.99 (0.92–1.07)	1.76 (1.22–2.55)	0.98 (0.93–1.02)	1.08 (0.91–1.29)
Unplanned cesarean	1.35 (1.33–1.37)	1.32 (1.18–1.48)	1.25 (1.24–1.27)	1.27 (1.21–1.32)
Planned cesarean	1.21 (1.19–1.22)	1.17 (1.06–1.29)	1.18 (1.17–1.19)	1.18 (1.14–1.22)
Placental abruption	1.95 (1.81–2.10)	1.62 (0.92–2.84)	1.72 (1.61–1.84)	1.75 (1.40–2.17)
Postpartum hemorrhage	1.46 (1.40–1.52)	1.47 (1.16–1.88)	1.37 (1.32–1.42)	1.46 (1.30–1.63)
Cerebrovascular accident	6.24 (4.78–8.15)	—§	2.70 (2.31–3.15)	3.04 (1.78–5.19)
Pulmonary edema	7.49 (5.29–10.61)	—§	4.64 (3.49–6.16)	—§
Acute renal failure	9.82 (8.44–11.43)	9.03 (4.20–19.43)	4.46 (3.82–5.21)	6.51 (4.25–9.96)
Severe obstetric morbidity	3.16 (2.96–3.38)	4.22 (2.85–6.24)	2.30 (2.16–2.44)	3.08 (2.55–3.72)

* Models include age, educational attainment, payment method for birth hospitalization, body mass index, obstetric history, delivery year, and state.

† Includes people who identified as multiple races or as a race not classified with the other racial groups, defined previously in the data sets by state health departments.

‡ Each mode of birth was compared with spontaneous vaginal birth.

§ Model did not converge due to small sample sizes.

with both unplanned and planned cesarean births, in contrast to a study of births at two hospitals in England.[8] We also found chronic hypertension to have a stronger association with cardiovascular anomalies than other anomalies, in agreement with a study that used Medicaid birth data from 2000 to 2007.[9] In addition, we calculated PAR percentages to quantify the potential effect of chronic hypertension in pregnancy at the population level. We estimated that chronic hypertension contributed to nearly one-quarter of all preeclampsia with severe features or eclampsia cases and a substantial percentage of cerebrovascular accidents (8.4%), pulmonary edema cases (10.7%), and acute renal failure cases (13.6%). These estimates suggest that chronic hypertension contributes to the population burden of these multifactorial outcomes. The latter severe complications are likely attributable to uncontrolled, severe hypertension,[15] which underscores the need for better prevention and management of chronic hypertension to reduce the rates of severe pregnancy-related complications in the United States.

Several limitations should be considered when interpreting the results of this study. The linked data set of vital statistics and hospitalization discharge data provided information on measured demographic factors, diagnoses, and procedures with overall high validity,[28–31] but did not include information on blood pressure or medication use. Diagnoses and procedures were also only available from the hospitalization for birth, and have been shown to be underreported.[28] Further, previously undiagnosed chronic hypertension can be missed during routine prenatal care because of the normal decreases in blood pressure during the first and second trimesters of pregnancy.[15] Therefore, chronic hypertension cases in this study included an unknown range of severity and treatment access, and the reported prevalence of chronic hypertension is likely an underestimate. If

Table 4. Adjusted Risk Ratios* and 95% CIs for Associations Between Chronic Hypertension and Neonatal Outcomes, Stratified by Racial–Ethnic Group

Outcome	American Indian or Alaska Native	Asian	Black	Latino	Native Hawaiian or Other Pacific Islander	White	Multiracial or Other†
Stillbirth	—‡	2.36 (1.81–3.09)	1.97 (1.62–2.40)	1.97 (1.71–2.26)	3.77 (1.75–8.12)	1.84 (1.52–2.23)	1.11 (0.55–2.23)
SGA (birth weight less than the 10th percentile)	1.96 (1.62–2.38)	1.66 (1.60–1.72)	1.34 (1.31–1.37)	1.72 (1.68–1.77)	1.75 (1.40–2.18)	1.59 (1.55–1.63)	1.54 (1.43–1.66)
Gestational age at birth (wk)§							
23–27	4.99 (2.62–9.51)	4.21 (3.61–4.91)	2.89 (2.70–3.09)	4.15 (3.84–4.48)	7.13 (4.19–12.11)	3.38 (3.13–3.64)	3.19 (2.53–4.01)
28–31	6.74 (4.62–9.82)	5.77 (5.22–6.36)	3.58 (3.39–3.77)	5.29 (5.01–5.59)	6.21 (4.09–9.45)	3.90 (3.71–4.10)	4.85 (4.15–5.66)
32–36	2.56 (2.21–2.96)	2.85 (2.74–2.97)	2.19 (2.15–2.24)	2.67 (2.61–2.72)	2.69 (2.29–3.15)	2.25 (2.21–2.29)	2.52 (2.37–2.67)
37–42	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Birth weight (g)§							
Less than 1,500	5.15 (3.50–7.59)	6.00 (5.54–6.51)	3.44 (3.30–3.58)	5.19 (4.96–5.43)	7.03 (4.97–9.96)	4.04 (3.87–4.22)	4.28 (3.75–4.90)
1,500–2,499	3.21 (2.70–3.81)	3.01 (2.90–3.13)	2.13 (2.09–2.18)	2.99 (2.92–3.07)	2.98 (2.44–3.64)	2.41 (2.36–3.46)	2.78 (2.60–2.97)
2,500 g or greater	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Congenital anomaly§							
Cardiovascular	2.26 (1.60–3.20)	2.32 (2.12–2.53)	1.86 (1.78–1.95)	2.20 (2.10–2.30)	1.92 (1.33–2.78)	1.89 (1.81–1.96)	2.06 (1.81–2.35)
Other anomaly	1.65 (1.19–2.29)	1.44 (1.33–1.56)	1.30 (1.24–1.36)	1.57 (1.50–1.64)	1.61 (1.17–2.22)	1.28 (1.24–1.33)	1.37 (1.21–1.55)
None	Ref	Ref	Ref	Ref	Ref	Ref	Ref

SGA, small for gestational age; Ref, reference.
* Models include age, educational attainment, payment method for birth hospitalization, body mass index, obstetric history, delivery year, and state.
† Includes people who identified as multiple races or as a race not classified with the other racial groups, defined previously in the datasets by state health departments.
‡ Model did not converge due to small sample sizes.
§ For multinomial outcomes, each outcome was compared with the most common outcome (gestational age 37–42 weeks, birth weight 2,500 g or greater, and no congenital anomaly).

underreporting of chronic hypertension and its severity was more common in racial–ethnic minoritized groups, associations with outcomes within those groups would also be underestimates–potentially resulting in lower estimates of racial–ethnic disparities. In this case, greater racial–ethnic disparities may actually exist as a result of chronic hypertension.

Our study used self-reported race and ethnicity, which is the gold standard reporting method and overall has high validity (eg, sensitivity of 94% or more).[30,31]

Table 5. Adjusted Population Attributable Risk Percentages* and 95% CIs for Associations Between Chronic Hypertension and Obstetric Outcomes, Stratified by Racial–Ethnic Group

Outcome	American Indian or Alaska Native	Asian	Black	Latino	Native Hawaiian or Other Pacific Islander	White	Multiracial or Other†
Preeclampsia with severe features or eclampsia	23.7 (20.9–26.5)	26.3 (25.6–27.1)	28.2 (27.8–28.7)	19.8 (19.5–20.2)	26.3 (23.2–29.2)	20.5 (20.2–20.8)	23.6 (22.5–24.6)
Mode of birth‡							
Operative vaginal	−0.7 (−1.3 to 0.03)	0.04 (−0.04 to 0.1)	−0.4 (−0.6 to −0.1)	−0.01 (−0.1 to 0.1)	1.6 (0.3–2.9)	−0.03 (−0.1 to 0.03)	0.1 (−0.1 to 0.4)
Unplanned cesarean	0.7 (0.2–1.1)	0.6 (0.5–0.6)	1.6 (1.5–1.7)	0.7 (0.6–0.7)	1.1 (0.6–1.6)	0.7 (0.6–0.7)	0.8 (0.6–0.9)
Planned cesarean	0.9 (0.5–1.2)	0.5 (0.5–0.6)	1.2 (1.1–1.3)	0.5 (0.4–0.5)	0.7 (0.2–1.1)	0.5 (0.5–0.5)	0.6 (0.5–0.7)
Placental abruption	0.7 (−1.3 to 2.7)	1.3 (1.0–1.7)	3.3 (2.8–3.9)	1.5 (1.3–1.7)	1.8 (−0.8 to 4.3)	1.3 (1.1–1.5)	1.6 (0.8–2.4)
Postpartum hemorrhage	0.2 (−0.7 to 1.1)	9.2 (7.4–10.9)	2.6 (2.2–2.9)	0.8 (0.7–0.9)	1.5 (0.4–2.7)	0.8 (0.7–0.9)	1.1 (0.7–1.5)
Cerebrovascular accident	—§	8.3 (4.0–12.5)	15.5 (12.7–18.2)	7.7 (5.6–9.7)	—§	5.3 (4.1–6.5)	5.7 (1.4–9.9)
Pulmonary edema	—§	5.9 (2.2–10.0)	14.3 (8.6–19.7)	12.1 (8.3–15.7)	—§	9.2 (6.4–11.9)	—§
Acute renal failure	—§	10.8 (8.0–13.5)	18.8 (16.3–21.3)	15.4 (13.5–17.2)	27.3 (10.5–41.1)	8.9 (7.4–10.4)	15.1 (9.3–20.5)
Severe obstetric morbidity	5.0 (1.1–8.8)	3.7 (3.0–4.3)	9.0 (8.2–9.8)	3.9 (3.6–4.3)	11.6 (6.4–16.5)	3.2 (2.9–3.5)	5.5 (4.2–6.9)

* Models include age, educational attainment, payment method for birth hospitalization, body mass index, obstetric history, delivery year, and state.
† Includes people who identified as multiple races or as a race not classified with the other racial groups, defined previously in the datasets by state health departments.
‡ Each mode of birth was compared with spontaneous vaginal birth.
§ Model did not converge due to small sample sizes.

Table 6. Adjusted Population Attributable Risk Percentages* and 95% CIs for the Associations Between Chronic Hypertension and Neonatal Outcomes, Stratified by Racial–Ethnic Group

Outcome	American Indian or Alaska Native	Asian	Black	Latino	Native Hawaiian or Other Pacific Islander	White	Multiracial or Other†
Stillbirth	—‡	3.5 (2.0–5.0)	6.0 (3.9–8.1)	2.1 (1.6–2.7)	10.2 (0.9–18.6)	2.2 (1.3–3.1)	0.5 (−2.8 to 3.6)
SGA (less than the 10th percentile)	2.0 (1.2–2.7)	0.9 (0.8–1.0)	1.5 (1.4–1.6)	0.9 (0.9–1.0)	1.7 (0.8–2.5)	1.0 (1.0–1.1)	1.1 (0.8–1.3)
Gestational age at birth (wk)§							
23–27	7.9 (2.5–12.9)	5.7 (4.7–6.7)	8.3 (7.6–9.0)	4.8 (4.4–5.3)	13.4 (6.7–19.8)	4.7 (4.2–5.1)	5.1 (3.6–6.6)
28–31	12.0 (7.7–16.2)	7.6 (6.9–8.4)	10.6 (10.0–11.3)	6.1 (5.8–6.5)	11.2 (6.7–15.6)	5.2 (4.9–5.5)	7.7 (6.4–9.0)
32–36	3.9 (3.0–4.8)	3.0 (2.8–3.1)	5.8 (5.6–6.0)	2.7 (2.6–2.8)	4.5 (3.5–5.6)	2.6 (2.5–2.7)	3.5 (3.2–3.8)
Birth weight§							
Less than 1,500 g	9.1 (5.5–12.5)	8.5 (7.8–9.1)	10.5 (10.0–11.0)	6.5 (6.2–6.8)	13.8 (9.4–18.0)	5.9 (5.6–6.2)	7.0 (6.0–8.1)
1,500–2,499 g	4.8 (3.7–5.9)	2.8 (2.7–3.0)	5.1 (4.9–5.2)	2.8 (2.7–2.9)	4.8 (3.4–6.0)	2.6 (2.5–2.7)	3.6 (3.2–3.9)
Congenital anomaly§							
Cardiovascular	3.4 (1.4–5.4)	2.5 (2.1–2.9)	4.9 (4.5–5.4)	2.2 (2.0–2.4)	3.1 (0.8–5.4)	2.0 (1.9–2.2)	2.9 (2.2–3.5)
Other anomaly	1.7 (0.3–3.0)	0.7 (0.6–0.9)	1.6 (1.3–1.9)	1.0 (0.9–1.1)	1.9 (0.3–3.5)	0.6 (0.5–0.7)	0.9 (0.5–1.3)

* Models include age, educational attainment, payment method for birth hospitalization, body mass index, obstetric history, delivery year, and state.

† Includes people who identified as multiple races or as a race not classified with the other racial groups, defined previously in the datasets by state health departments.

‡ Model did not converge due to small sample sizes.

§ For multinomial outcomes, each outcome was compared with the most common outcome (gestational age 37–42 weeks, birth weight of 2,500 g or greater, and no congenital anomaly).

However, a prior validation study found sensitivity for American Indian or Alaska Native to be only 54%, suggesting misclassification that could bias results for this population.[30] We were able to adjust analyses for BMI, which is not readily available in discharge data. Weight and height information in vital statistics data can be self-reported, and a systematic review found people underreport their prepregnancy weight by 0.3–3 kg; this reporting error was found to largely not bias associations with outcomes.[32] The study data contained limited information on resource utilization, and future research is needed to estimate the burden of chronic hypertension in pregnancy on the U.S. health care system.

We used PAR percentages as a way to assess the population-level effect of chronic hypertension in pregnancy. These estimates, however, are not equivalent to the potential effect of a public health intervention on chronic hypertension in pregnancy because PAR percentages make the unrealistic assumptions that an intervention could eradicate an exposure and doing so would not affect other exposures.[33] Future work that considers chronic hypertension along with co-exposures as potential drivers of the incidence of and disparities in adverse pregnancy outcomes would be a valuable extension of the current study. In addition, this was an observational study using routinely collected health data; therefore, residual confounding is likely and causality cannot be established. The study population drew from five states in the United States, but generalizability to other regions was not assessed.

In conclusion, this analysis found that chronic hypertension in pregnancy substantially increases the risk of many adverse obstetric and neonatal outcomes, particularly preeclampsia with severe features or eclampsia, cerebrovascular accidents, acute renal failure, pulmonary edema, very preterm birth, and very low birth weight. Further, chronic hypertension contributes to racial–ethnic disparities in complications during birth hospitalization for people who are Black or Native Hawaiian or Other Pacific Islander.

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